The two faces of network dynamics

Evolving network models describe the dynamics (assembly, evolution) OF networks by the addition/removal of nodes and edges.

It is possible to have network dynamics even if there are no node/edge additions/removals, i.e. the network is fixed. This can be called dynamics ON the network.

One scenario for dynamics on a network is the spreading of disease or of information on a social/contact network.

Another scenario is when the nodes represent species and the edges represent interactions that lead to changes in the species abundance, e.g. predator-prey interactions or chemical reactions.

Node state (status)

In many networks node attributes can change in time due to the interactions represented by edges. E.g.

- the abundance of molecules in a chemical reaction network
- the health status of individuals in a disease contact network

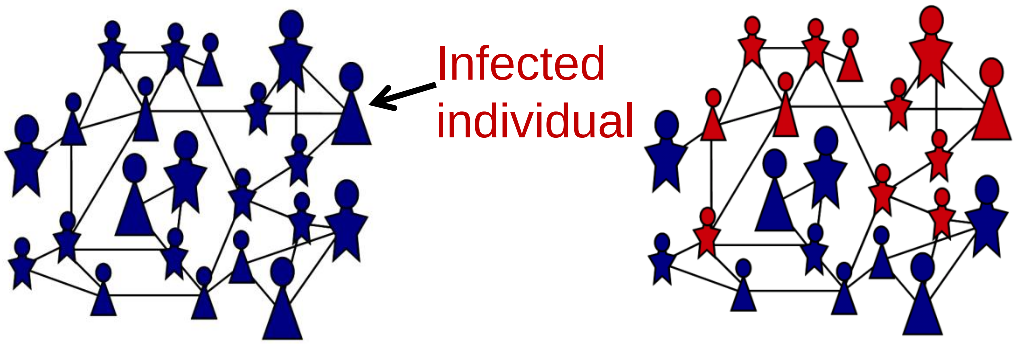
For these networks it is not enough to specify the nodes and edges, we also need to define a node state (e.g. a continuous variable, or a discrete category).

Each node's state is determined by the states of the nodes adjacent to it; in directed networks the orientation of the edges should be toward the regulated node. The functional form of the dependence varies from application to application.

Epidemic spreading on contact networks

Human contact network

Nodes: individuals
Edges: disease specific physical contacts



Any infected neighbor of a susceptible individual can infect it.

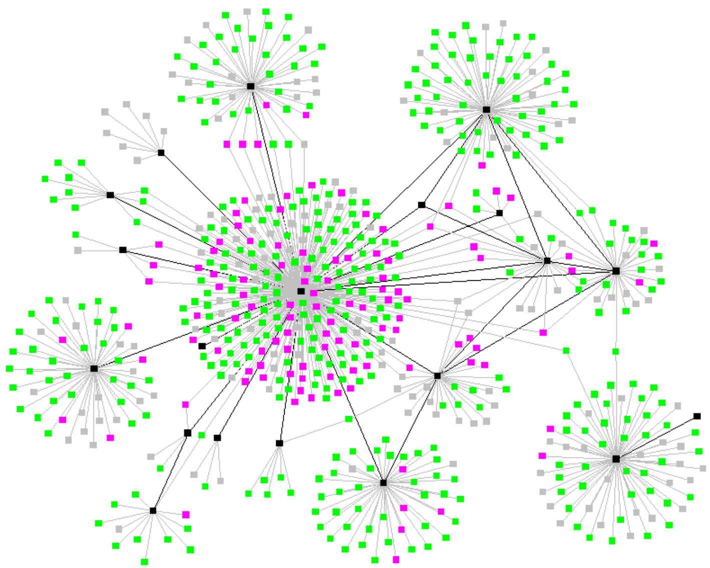
The infection can propagate through the network, and cause an epidemic.

Questions asked:

- Will an epidemic occur?
- How do we control the spread?

Network model of epidemic spread

• Assume that the contact network is known.
• Assume that there is a constant disease transmission probability T along each edge.
• Nodes can be susceptible or infected. Assume that there is a single infected node at the beginning.



• The disease spreads with probability T to each of the first neighbors of the infected node, then to the second, ….

• Epidemic: the infection spreads to the whole network or a sizable fraction of it

Q. Is it possible to foresee whether an epidemic can occur without simulating the spreading process?

Reduce to a static network problem

- At first, each node (individual) is susceptible

- Mark (or occupy) each edge in the social network with probability T. Disregard the other edges.

- The ultimate size of an outbreak would be the set of nodes that can be reached from an infected node by traversing marked edges.

- Thus, we only need to determine the sizes of the connected components formed by marked edges. We know that infection of any of the nodes in a component will cause an outbreak equal to the size of the component.

- If a marked component is a giant connected component, an infection of any of the nodes in that cluster will cause an epidemic.

Epidemics on a general random network

Start with a random network with a given degree distribution P(k).

Mark edges with probability T. Disregard the unmarked edges.

Expectation: if $T>T_c$ there will exist a large connected component of marked edges.

T_c depends on P(k)

There is a connection between epidemics on graphs and breakdown of graphs when edges are deleted (Q: which two parameters are equivalent?)

Result:

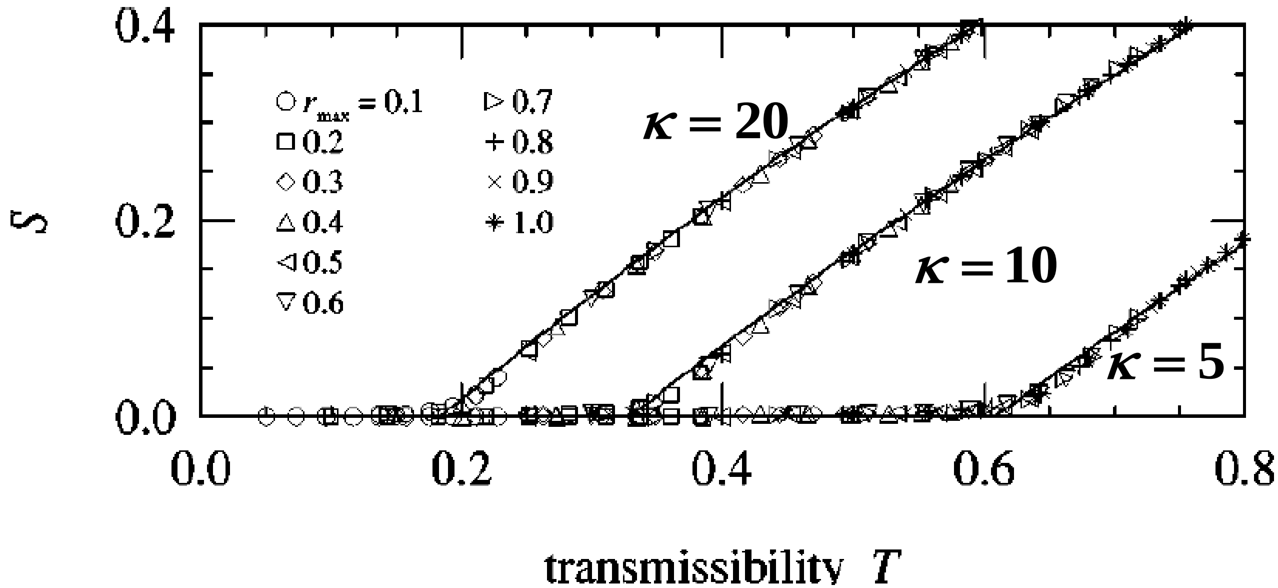
$$T_c = \frac{1}{\langle k^2 \rangle / \langle k \rangle - 1}$$

Example: scale-free network

Scale-free

with cutoff at k=κ

$$P(k) \approx k^{-\alpha} e^{-k/\kappa}$$



T_c decreases with **κ**

$$\lim_{\kappa \to \infty} T_c = 0$$

Any infection leads to epidemics in infinite scale-free networks with no cutoff.

M. E. J. Newman, Phys. Rev. E 66, 016128 (2002)

Ex.

Consider a population through which an epidemic just swept. Shortly thereafter there is a second outbreak of the same disease. Assume that individuals infected in the first outbreak cannot contract the disease again (they are immune).

How would you estimate what fraction of the population will be infected in the second outbreak ?

Dynamics of epidemic spreading on a statistically homogeneous network

Assume that individuals can be susceptible or infected. Define the fraction of individuals in each category.

$$s = \frac{S}{N}, i = \frac{I}{N}, \; s + i = 1$$

Define the transmission rate per edge, λ

The fraction of infected neighbors of a susceptible node is $\langle k \rangle i$

$$\frac{ds}{dt} = -\lambda \langle k \rangle is, \qquad \frac{di}{dt} = \lambda \langle k \rangle is$$

Initial spread (when s~1): $i(t) \approx i_0 e^{t/\tau_H}, \quad \tau_H = 1 / \lambda \langle k \rangle$

τ_H - time scale governing the growth of the infection

Heterogeneous network

Focus on nodes with given degrees

$$i_k = \frac{I_k}{N_k}, \; N_k = NP(k), \;\; s_k = 1 - i_k$$

Assumptions: nodes with degree *k* are equivalent

the fraction of infected nodes that are neighbors of a susceptible node with degree *k* equals $k\Theta$

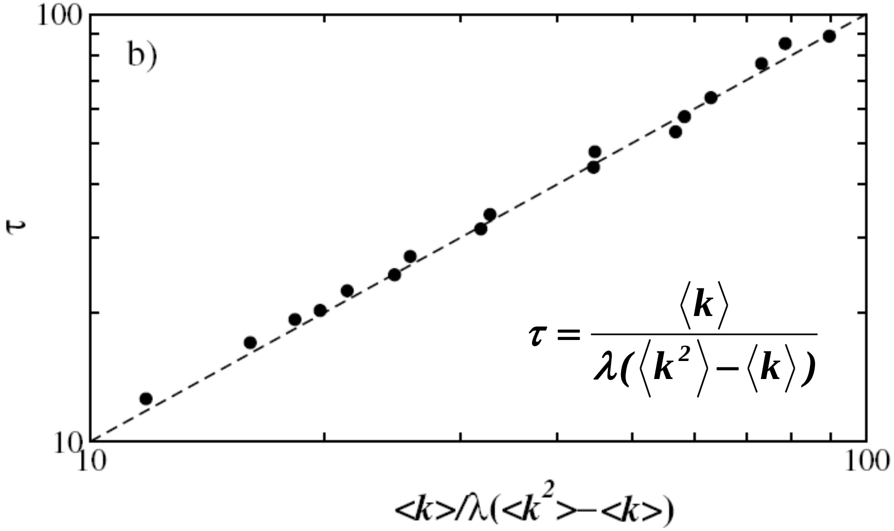
Θ - the density of infected neighbors of a vertex

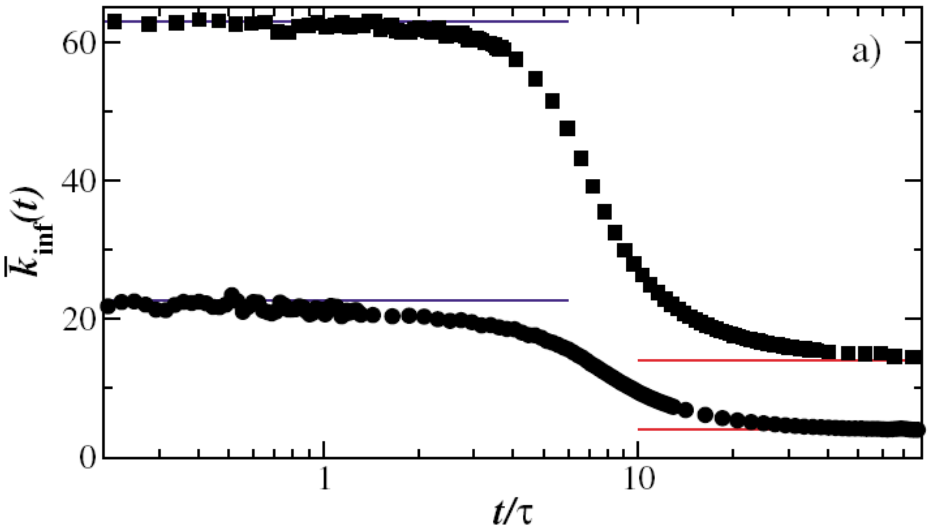
$$\frac{di_k(t)}{dt} = \lambda\, k\, s_k(t)\Theta(t)$$

Initial spread:

$$i(t) = i_0\left[1 + \frac{\langle k\rangle^2 - \langle k\rangle}{\langle k^2\rangle - \langle k\rangle}\left(e^{t/\tau} - 1\right)\right] \qquad \tau = \frac{\langle k\rangle}{\lambda(\langle k^2\rangle - \langle k\rangle)}$$

Simulation results agree with theory





The timescale of the initial spreading process agrees with calculation.

The average degree of newly infected nodes $\langle k^{inf} \rangle$ is high at the beginning of the spreading process.

Barthelemy et al, Phys. Rev. Lett. 92, 178701 (2004)

How to control the epidemic?

- Transmission-reducing interventions: face masks, gloves, washing hands – may reduce the transmission rate below the epidemic-causing critical rate

- Contact-reducing interventions: quarantining a patient, closing schools – make the network sparser, may increase the critical transmission rate

- Vaccinations: remove nodes from the network

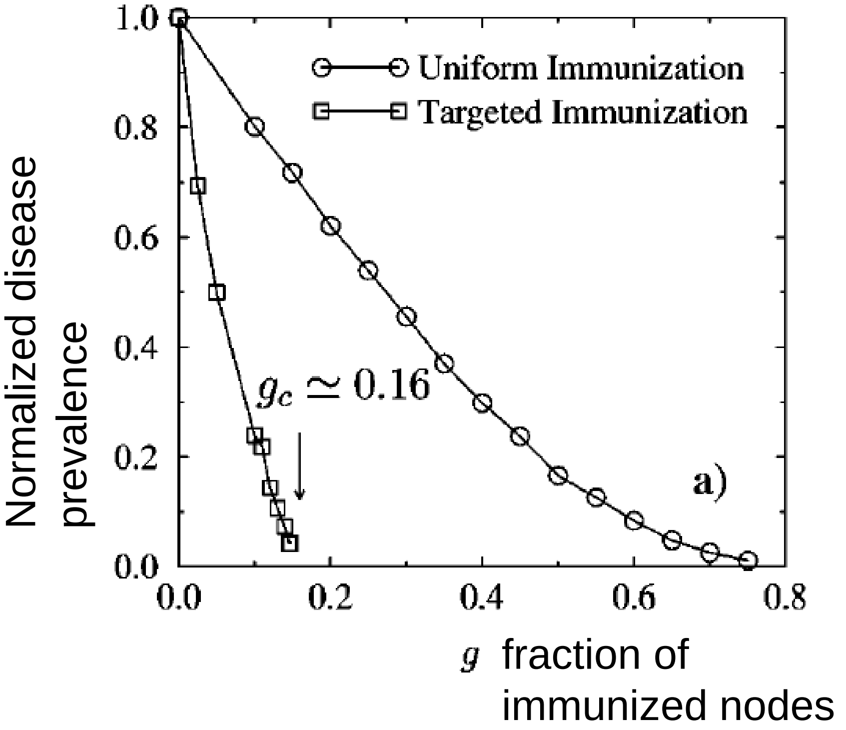
- Q: Who should be vaccinated for most effective control?

Vaccination strategies in scale-free networks

SIS model, contact network constructed by BA model.

Targeted immunization – immunize highest degree nodes.

Uniform immunization ineffective in heterogeneous networks, targeted immunization needed



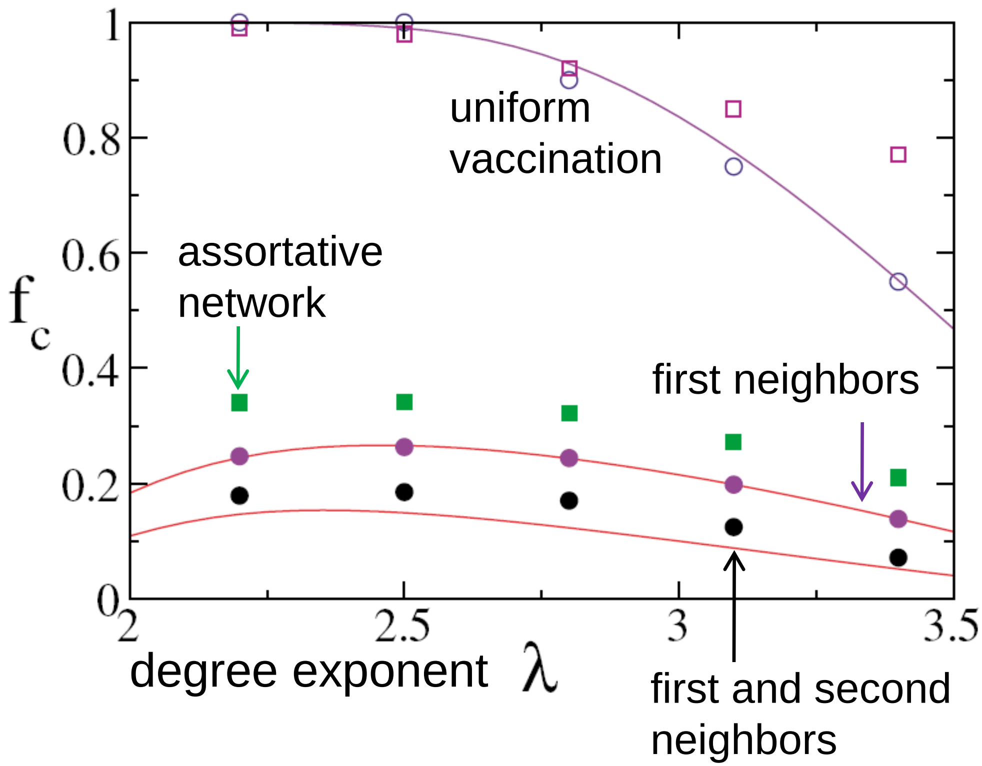
R. Pastor-Satorras and A. Vespignani, Phys. Rev. E 65, 036104 (2002)

A local method for vaccination effective in scale-free networks

Contact network described by scale-free random graph

Immunization strategy: select a node randomly, immunize a randomly selected neighbor of it.

Use theory and simulations to determine the critical immunization fraction for each transmissibility value



R. Cohen et al, Phys. Rev. Lett 91, 247901 (2003)

Rumor and information spreading

The models are similar to epidemic models, with a translation of state terms:

Ignorant (has not yet learned the new information)
Spreader
Stifler (aware of the new info but no longer spreading it)

Important dissimilarities with disease spreading:

– The spread of information is an intentional act
– Acquiring information is not a passive process, memory may play a role
– The transition to a stifler state is not usually spontaneous

For more info see Chapter 10 in Dynamical Processes on Complex Networks (Barrat, Barthelemy, Vespignani, 2008)

A model of cascading processes on random networks

Assume a random network with a given degree distribution
Two node states (e.g. ignorant and spreader); at the beginning all nodes but one are ignorant
Each node becomes a spreader if the fraction of its neighbors who are spreaders is larger than a threshold.

Early adopter: a node that will become spreader if one of its neighbors is a spreader.

A global cascade is possible if the early adopters form a giant connected component of the network. Formally, $\sum_k k(k-1)\rho_k P(k) > \langle k \rangle$

where ρ_k is the probability that a degree-k node is an early adopter.

Both the heterogeneity of the network and of the adoption thresholds plays a role in the adoption of new innovations.

D. Watts, PNAS 99, 5766 (2002)